Water and electrolytes + acid base balance:

1. A 25 year old army recruit was feeling weakness after a 10 km race. He is confused and his blood pressure is 110/80. Labs showed serum sodium of 118 with all other tests being normal. What would be the most likely cause of low sodium in this patient?
   A. Cerebral salt wasting
   B. Salt wasting nephropathy
   C. Inappropriate ADH secretion
   D. Dilutional hyponatremia
   E. Excessive sweating

Ans: Dilutional hyponatremia

Explanation: Exercise induced hyponatremia or dilutional hyponatremia or water intoxication occurs when water is taken more than 1000 ml per hour at which level kidneys ability to excrete water becomes saturated and free water starts accumulating and causes dilutional hyponatremia. Prevention is the best treatment for this and athletes should be advised to take water as required and use salt containing water if they have to exercise for more time.

2. Which of the following is the best initial treatment for a patient with euvolemic hyponatremia who is asymptomatic?
   A. Furosemide
   B. Conivaptan
   C. Free water restriction
   D. Hypertonic saline
   E. 5% dextrose

Ans: Free water restriction

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Explanation: For normovolemic hyponatremia, free water restriction is generally the treatment of choice (< 1L/day). Hypertonic saline has no role in asymptomatic patients and used in patients who have neurological complications. Treat patients with hypervolemic hyponatremia with salt and fluid restriction plus loop diuretics and correction of underlying condition. The use of conivaptan which is V2 receptor antagonist is considered in the treatment of hypervolemic hyponatremia. 5% dextrose should be avoided as it will worsen the hyponatremia.

3. Which of the following is a characteristic feature of SIADH?
   A. Urinary K of 30 mEq/L
   B. Urinary sodium of 10 mEq/L
   C. Urine osmolality of > 100 mOsm/kg
   D. Low serum cortisol
   E. Hypovolemia

Ans: Urine osmolality of > 100 mOsm/kg

Explanation: Barrter and Schwartz criteria for diagnosis of SIADH:

- Decreased serum osmolality (<275 mOsm/kg)
- Increased urine osmolality (>100 mOsm/kg)
- Euvolemia
- Increased urine sodium (>20 mmol/L)
- No other cause for hyponatremia (no diuretic use and no suspicion of hypothyroidism, cortisol deficiency, marked hyperproteinemia, hyperlipidemia or hyperglycemia).

4. A 28 year old male is brought in the emergency after collapsing at a night party with an episode of generalized tonic clonic fits. Examination showed GCS of 10 with blood pressure of 160/100. Labs were done which showed serum sodium of 116. Which of the following should be considered in this patient as the cause of above presentation?
   A. Cerebral salt wasting syndrome
   B. Beer potomania
   C. Ecstasy poisoning
   D. Renal salt wasting
   E. Viral encephalitis

Ans: Ecstasy poisoning

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Explanation: Ecstasy poisoning should be considered in a patient with hyponatremia and some night party attendance. High blood pressure is also a clue here. Ecstasy (MDMA) is a common party drug as it is hallucinogenic and also has amphetamine like properties. Hyponatremia is associated with Ecstasy use either because of an inappropriate secretion of antidiuretic hormone or because of sodium loss through excessive sweating during dancing combined with electrolyte-free water intake. Beer potomania occurs in chronic alcoholic patients who take excessive alcohol for prolonged time.

5. A 41 year old man presents to the emergency room with a 5 days history of nausea and vomiting. Physical examination showed weight 70kg with decrease skin turgor and there is postural hypotension. Lab studies showed serum sodium 116 meq/L, serum creatinine 1.5mg/dl, urine osmolality 1000 mOsm/kg and urine sodium concentration of 4 meq/L. Which of the following should be the most appropriate initial treatment in the emergency room?

   A. Free water restriction
   B. 3% Hypertonic saline
   C. 0.9% saline 2 L
   D. 0.45% saline 100ml/h
   E. 5% dextrose saline

Ans: 0.9% saline 2 L

Explanation: This patient has hypovolemic hyponatremia due to vomiting and labs also showed high urine osmolality with low urinary sodium, so the best initial treatment in this patient should be to give plasma expander. The best fluid for this purpose is 0.9% saline 2 L initial bolus followed by infusion until the postural hypotension resolved and then according to the clinical indications. Hypertonic and hypotonic fluids are not indicated in hypovolemic hyponatremia. Free water restriction in a patient with postural hypotension is not a good option here and can only be used in euvolemic hyponatremia with urinary sodium more than 40.

6. A 68 year old man is found to have hyponatremia during a routine visit. He is on no medications but he smoked 1 pack of cigarettes per day for 25 years. On physical examination, he is alert and oriented and blood pressure is 125/80 mmhg and there is no edema and his chest is clear to auscultation. Lab studies show serum sodium 125 mEq/L, creatinine 1.3mg/dl, urine sodium 85 mEq/L and urine osmolality 600 mosm/kg. The most appropriate treatment in this patient is:

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A. 3% saline
B. 0.9% saline
C. Free water restriction
D. Desmopression
E. Demeclocycline

Ans: Free water restriction

Explanation: This patient who is a smoker may have developed lung cancer which is secreting ADH inappropriately. The best treatment for inappropriate ADH secretion is free water restriction. This patient has clinically normal ECF volume, elevated urinary sodium and urine osmolality greater than 100 which are all consistent with SIADH.

7. A 67 year old male presented with history of progressive loss of consciousness for 5 days. On examination, patient is unconscious and his weight is 50kg, blood pressure is 90/60 mmhg. His skin turgor is decreased. Labs showed serum sodium 167 meq/L, creatinine 1.5 mg/dl, and urine sodium 8meq/L and urine osmolality of 1050 mosm/kg. The most appropriate initial treatment for this patient should be:

A. 0.45% saline
B. 5% dextrose
C. 3% saline
D. 0.9% saline
E. Desmopression

Ans: 0.9% Saline

Explanation: This patient is presented with evidence of water depletion more than sodium depletion leading to hypernatremia due to dehydration. The initial goal should be to restore the ECF volume to restore the organ perfusion. The best initial treatment should be an isotonic solution to expand the plasma. 0.9% saline is the best choice here. Also, the initial serum osmolality in hypernatremia is much more than the osmolality of 0.9% saline so, initially isotonic saline is relatively hypotonic to the serum and will lower the serum sodium gradually. Rapidly correcting hypernatremia with hypotonic solution like 5% dextrose will cause pontine demyelination. In a 50 kg male, initial treatment with 1-1.5 L of isotonic saline is required. Once the ECF volume is restored with isotonic saline, it should be followed by 5% dextrose solution after estimation of water deficit.

8. A 27 year old female is presented with history of polyuria and polydipsia for 3 months with normal HBA1C. Serum sodium is 149 meq/L. Urine osmolality is 80 mosmol/kg and it remains 90 mosmol/kg after 3 hours of water deprivation but increases to 650 mosmol/kg after S/C desmopression. The best treatment option for this patient is

A. Oral desmopression
B. Repeated 5% dextrose infusions
C. Hydrochlorothiazide
D. Intranasal desmopression
E. Demeclocycline

Ans: Intranasal desmopression

Explanation: This patient has central diabetes insipidus based on rise in urine osmolality after administration of desmopression. Treatment of central DI is directed towards replacing the ADH with

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desmopression. Intranasal route is more reliable than oral form due to variable oral absorption. Hydrochlorothiazide is used in Nephrogenic DI. Demeclocycline is used in SIADH.

9. A 55 year old male known case of diabetes mellitus on insulin presented with polyuria and excess thirst for 5 days and now complaining of weakness. Examination showed decrease skin turgor and weight of patient is 55kg. Patient was not using insulin for 1 week. Labs were done which showed serum sodium of 168meq/L. What will be the water deficit of this patient?

   A. 4.5 L
   B. 5.0 L
   C. 6.0 L
   D. 6.5 L
   E. 7.5 L

   Ans: 6.5 L

   Explanation: The total body water deficit in the hyperosmolar patient that needs to be replaced can be roughly estimated using the following formula:

   TBW deficit = correction factor x premorbid weight x (measured Na/140-1)

   For males correction factor is 0.6 and for females it is 0:5. By using this formula, the total body water deficit in this patient is 6.6 L which is approx. equal to 6.5 L of fluid that should be infused in this patient to correct the hypernatremia. This should be replaced slowly in 48-72 hours and plasma sodium should not be lowered more than 1 mmol/l in 1 hour and not more than 12 mmol /l in first 24 hours. To reduce serum sodium dextrose water is used or 0.45 % saline is used if blood sugar is high.

   - Hypernatremia with hypovolemia=> 0.9% saline initially followed by 5% dextrose to correct free water deficit
   - Hypernatremia with euvolemia => free water or 5% dextrose
   - Hypernatremia with hypervolemia => 5% dextrose plus loop diuretic

10. A 45 year old male is admitted due to CAP 2 days back. You received the call from your house officer that patient is becoming drowsy and has had 1 episode of generalized tonic clonic fits. Labs were done which showed serum sodium of 112. What should be the next best management step?

    A. Free water restriction
    B. 3% saline infusion
    C. 0.9% saline
    D. Do CT brain
    E. Start phenytoin infusion

    Ans: 3 % Saline infusion

    Explanation: This patient has developed acute hyponatremia due to SIADH secondary to community acquired pneumonia. As, the patient is symptomatic and there is neurological involvement, so the best treatment in this patient should be 3% hypertonic saline infusion.

11. A known case of type 1 diabetes mellitus patient presented in emergency with a diagnosis of diabetic ketoacidosis. His blood sugar at presentation was 650mg/dl. Serum sodium at presentation was 132 meq/L. What would be the corrected sodium in this patient?

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A. 140 meq / L
B. 135 meq/L
C. 125 meq / L
D. 130 meq / L
E. 145 meq / L

Ans: 140 meq / L

Explanation: The serum sodium result may be altered by pre-existing high glucose levels in patients with hyperglycemia. In the presence of abnormally high blood glucose levels, if there is no change in the total body water the observed low sodium levels are likely due to the hyponatremic response and should not be confused with established hyponatremia. The correction factor is to add 1.6 meq with each 100 mg of blood sugar higher than the normal or use this formula:

Sodium Correction = Measured sodium + 0.016 x (Serum glucose -100)

12. A 45 year old patient is admitted with a history of spontaneous aneurysmal subarachnoid hemorrhage 3 days back. Today, the sensorium of patient has deteriorated and patient looks dehydrated with decrease skin turgor. Blood pressure is also lower than at admission. CT brain has not shown new changes. Labs showed serum sodium 123 meq/L, urinary sodium 65 meq /L and urine osmolality 310 mosmol /kg. What is the most probable diagnosis in this patient?
    A. SI ADH secretion
    B. Cerebral salt wasting syndrome
    C. Vasospasm
    D. Pituitary apoplexy
    E. Reset osmostat

Ans: Cerebral salt wasting syndrome

Explanation: Hyponatremia after aneurysmal subarachnoid hemorrhage with hypovolemia and hypotension occurs due to cerebral salt wasting syndrome. This must be differentiated from SIADH which is a euvolemic state and treatment of both are opposite to each other. In cerebral salt wasting, fluid is given with salt intake, whereas in SIADH, water restriction is the best treatment. Pituitary apoplexy presents with new onset headache with circulatory collapse. Reset osmostat occurs in pregnancy.

13. A 24 year old male who is a known case of hypertriglyceridemia came for review of labs which showed serum triglycerides 860 mg/dl and total cholesterol 270 mg/dl. Serum sodium is 127 meq/L with normal serum and urine osmolality. Patient is taking fenofibrate, rosuvastatin and aspirin. What is the likely cause of hyponatremia in this patient?
    A. SIADH
    B. Rosuvastatin
    C. Aspirin
    D. Pseudohyponatremia
    E. Fenofibrate

Ans: Pseudohyponatremia

Explanation: Pseudohyponatremia is a laboratory abnormality that is defined by a serum sodium concentration of less than 135 mEq/L in the setting of a normal serum osmolality (280 to 300 mOsm/kg). Conversely, true hyponatremia is associated with low serum

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osmolality and should prompt evaluation for the presence of an additional abnormal solute that may be affecting the laboratory assessment. The finding of pseudohyponatremia is an artifact. The most common cause of pseudohyponatremia is due to severely elevated levels of cholesterol. In serum blood samples taken from patients with severe hypertriglyceridemia, the sample may appear overtly lipemic, hyper viscous, or discolored from the overwhelming presence of insoluble triglycerides. In the presence of an abnormally excessive level of additional solute, the ratio of solid to water in plasma is altered unpredictably, leading to inaccurate readings when sodium ions are measured indirectly.

14. You have received a call from surgery department for a patient who is feeling restlessness and weakness. He has laparotomy for strangulated hernia 5 days back. You have checked the morning labs which showed serum sodium of 121 mEq/L. What is the most probable cause of low sodium in this patient?
    A. SIADH
    B. Water intoxication
    C. GI loss
    D. Drug induced
    E. Renal salt wasting

Ans: Water intoxication

Explanation: Hypotonic fluids like dextrose water is commonly used postoperatively. Excess infusion of hypotonic fluids can cause hyponatremia because glucose of dextrose is metabolized and free water is equally distributed in all compartments and causes a dilutional effect on sodium level. SIADH can occurs in the first 24- 48 hours post-operative, but one of the common cause of post-operative hyponatremia is water intoxication due to overzealous hypotonic fluids infusion.

15. A patient is presented with weakness and muscles pain for 2 weeks. Labs results were done which showed serum phosphate of 1.5 mg/dl (normal > 2.5 mg). Fractional excretion of phosphate is 3%. What is the likely cause of Hypophosphatemia in this patient?
    A. Primary hyperparathyroidism
    B. Hypophosphatemic rickets
    C. Fanconi syndrome
    D. Uncontrolled DM
    E. Vitamin D deficient rickets

Ans: Vitamin D deficient rickets

Explanation: If the patient has hypophosphatemia, the next step should be to check for renal wasting of phosphate. This is done by calculating fractional excretion of phosphate. If it is less than 5%, it showed diminished supply or absorption. If more than 5%, it shows renal wasting. Causes of hypophosphatemia with less than 5% fractional excretion of phosphate are starvation, TPN, antacids and vitamin D deficient or resistant rickets. Primary and secondary hyperparathyroidism, uncontrolled DM, Fanconi syndrome and hypophosphatemic rickets all cause renal wasting of phosphate.

16. Which of the following is the cause of rapidly developing hypokalemia?
    A. Vomiting
    B. Thiazide diuretic
    C. Liddle syndrome
    D. RTA type 1

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E. RTA type 2

Ans: Vomiting

Explanation: Vomiting is associated with rapidly developing hypokalemia. There are three main reasons for developing hypokalemia in vomiting:

- Direct GI loss due to loss of gastric contents
- Acid loss due to loss of gastric acid leads to metabolic alkalosis which causes transcellular shift of K into the cells
- Urinary loss of K occurs because the first response of body is to preserve the ECF volume. The hypovolemia induced by vomiting causes an increase in aldosterone secretion which leads to loss of more potassium in the urine despite already low potassium.

17. A 35 year old male is presented with complaints of generalized body weakness for 2 months. Labs were done which showed serum sodium 139 mEq/ L, K 2.9 mEq /L, Cl 105 and HCO3 of 28. Blood pressure is 150/100 mmhg. Urinary Potassium is 45 mmol/day. What can be the possible cause of hypokalemia in this patient?
    A. Gitelman syndrome
    B. Renal tubular acidosis
    C. Diarrhea
    D. Liddle syndrome
    E. Diuretic abuse

Ans: Liddle syndrome

Explanation: In the approach to determine the cause of hypokalemia, the first step is to check urinary potassium, if it is more than 30 mmol/ day, it shows renal loss of potassium. If there is renal wasting then the next step is to check blood pressure. Low serum K, high urine K with high blood pressure occurs due to primary and secondary hyperaldosteronism, Cushing syndrome, apparent mineralocorticoid excess, liquorice ingestion and Liddle syndrome. To differentiate between these conditions, the next step is to measure serum aldosterone and plasma renin activity. High aldosterone with low PRA => Conn syndrome. High aldosterone with high PRA => secondary hyperaldosteronism due to renal artery stenosis. Low aldosterone with low PRA => Liddle syndrome and liquorice ingestion or apparent mineralocorticoid excess. Low aldosterone and high renin with high cortisol => Cushing syndrome.

Liddle's syndrome is a genetic disorder characterized by hypertension with hypokalemic metabolic alkalosis, hyporeninemia and suppressed aldosterone secretion. Liddle's syndrome is caused by mutations to subunits of the Epithelial Sodium Channel. Potassium-sparing diuretics such as amiloride and triamterene reduce ENaC activity, and in combination with a reduced sodium diet can restore normotension and electrolyte imbalance in Liddle's syndrome patients.

18. A 32 year female presented with muscle weakness. Labs were done which showed serum K 3.1, HCO3 29, pH 7.50, urine K 45 mmol/ day. Her BP is 168/105 mmhg. Plasma aldosterone and renin level are suppressed. What is the likely cause?
    A. Diuretic abuse
    B. Barter syndrome
    C. Conn syndrome
    D. Liquorice ingestion
    E. Renal artery stenosis

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Ans: Liquorice ingestion

Explanation: Hypokalemic metabolic alkalosis with renal K wasting associated with hypertension and suppressed aldosterone and renin level occur either in Liddle syndrome or liquorice ingestion. Liquorice is used for weight loss by homeopathics. It is also present as sweetener in confectionery. The active ingredient of liquorice, glycyrrhizic acid, and some of its metabolic by-products are inhibitors of 11β-hydroxysteroid dehydrogenase function. This can result in an apparent mineralocorticoid excess, a longer half-life of plasma cortisol and reduced plasma cortisone. Withdrawal of dietary liquorice, even after prolonged ingestion or ingestion of very large quantities, is associated with a sustained and complete resolution of the symptoms of induced apparent mineralocorticoid excess.

19. A 24 year old female presented with complaints of generalized muscular weakness and aches and pains for 3 months. Her BP is 110/70mmhg. Labs were done which showed serum K 2.7, HCO3 32, Cl 100, Na 135, and pH 7.52. Urinary K is 39 mmol / day. What can be the possible cause of low potassium in this patient?
    A. Renal tubular acidosis type 2
    B. Acetazolamide
    C. Furosemide abuse
    D. Laxative abuse
    E. Liquorice abuse

Ans: Furosemide abuse

Explanation: This patient is having hypokalemia with renal wasting with normal blood pressure. The next step in these patients should be to check the acid base status. Low K, high urinary K, normal blood pressure and acidosis is associated with RTA type 1 and type 2 and acetazolamide therapy, and with alkalosis the causes are thiazide or loop diuretics and Barter and Gitelman syndrome.

Laxative abuse will cause urinary potassium < 20. Liquorice ingestion is associated with hypertension.

20. A 14 year old male presented with failure to thrive with feeling weak all the times and passing excess urine with excess thirst for 2 years. Blood glucose checked many times is normal. Blood tests were done which showed serum K 2.6, HCO3 31, Cl 92, Na 131, and pH 7.49. Urinary electrolytes were done which showed higher levels of all the electrolytes. Blood pressure is normal. What is the possible diagnosis?
    A. Bartter syndrome
    B. Liddle syndrome
    C. Laxative abuse
    D. Chronic diarrhea
    E. Fanconi syndrome

Ans: Bartter syndrome

Explanation: Bartter syndrome is a rare genetic salt losing nephropathy characterized by renal K wasting with hypokalemia, metabolic alkalosis, high serum aldosterone and renin activity, normotension and resistance to the pressor effects of angiotensin II and JG cell hyperplasia. It is commonly presented in pediatric age group. The presenting features are failure to thrive, vomiting, polyuria, and dehydration. Disproportionate urinary wasting of sodium, potassium, and chloride are also seen. The diagnosis is confirmed by elevated serum levels of both renin and aldosterone with normotension. There is a defect in prostaglandins homeostasis which leads to elevated PGs level and

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peripheral vasodilation and lack of response to pressors. Indomethacin or ibuprofen therapy resulted in marked improvement.

Liddle syndrome is associated with hypertension, Fanconi syndrome is associated with acidosis, laxative abuse, and chronic diarrhea are associated with low urinary potassium.

21. A 15 year old male is diagnosed as having hypokalemic metabolic alkalosis with normal blood pressure. His urinary potassium is also high. You are suspecting Bartter or Gitelman syndrome as the cause. Which test you will order to differentiate between these two conditions?
    A. Serum magnesium
    B. Urinary chloride
    C. Urine calcium
    D. Aldosterone: PRA ratio
    E. Urinary sodium

Ans: Urinary calcium

Explanation: Both Bartter syndrome and Gitelman syndrome have same clinical manifestations of hypokalemic metabolic alkalosis, salt wasting with high urinary sodium and chloride with hypomagnesaemia. The most critical diagnostic step in differentiating between these two conditions is measurement of urinary calcium level. In Bartter syndrome it would be high and urine calcium/creatinine ratio would be more than 0.20 or more than 300 mg/day and in Gitelman syndrome it would be less than 0.15 or less than 50 mg/day. This differentiation is important because Bartter syndrome can be managed with NSAIDs whereas NSAIDs are ineffective in Gitelman syndrome. The mainstay of treatment for Gitelman syndrome is a high salt diet with oral potassium and magnesium supplements. Mg should be given in small frequent doses to avoid diarrhea which will worsen the hypokalemia.

22. A 26 year old female presented with complaints of muscle cramps with general body weakness for 2 months. Labs were done which showed serum K 2.6, HCO3 20, Cl 109 and Na 133. Urinary Potassium is 10 mmol/ day. Her BP is 115/85 mmhg. What can be the probable cause for the symptoms of this patient?
    A. Diuretic abuse
    B. Gitelman syndrome
    C. Acetazolamide abuse
    D. Laxative abuse
    E. Renal Tubular acidosis

Ans: Laxative abuse

Explanation: This patient is having hypokalemic metabolic acidosis with normal anion gap and low urinary potassium. The most common cause of hypokalemic non anion gap metabolic acidosis is diarrhea. Other possibility is laxative abuse for weight loss. Gitelman syndrome and diuretic abuse causes hypokalemic metabolic alkalosis with high urinary potassium. Acetazolamide and renal tubular acidosis is associated with hypokalemic metabolic acidosis with high urinary potassium.

23. A 24 year old female is diagnosed with new onset hypertension presented with headache. Further testing showed hypokalemia and patient was subsequently diagnosed as a case of Liddle syndrome. Which of the following is the preferred antihypertensive in this patient?
    A. Lisinopril
    B. Losartan
    C. Spironolactone

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D. Amiloride
E. Nifedipine

Ans: Amiloride
Explanation: Liddle syndrome is associated with gain of function mutation in epithelial sodium channels with overstimulation of this channel leading to hypokalemia and hypertension with depressed aldosterone and renin activity. The drug of choice is Amiloride because it is an inhibitor of ENaC. Spironolactone is ineffective in this condition because it is a receptor inhibitor and aldosterone levels are already suppressed. Other conventional antihypertensives are also ineffective.

24. A 27 year old male is presented with progressive weakness of all four limbs for 3 days. Examination showed flaccid paraparesis. Serum electrolytes were done which showed serum potassium of 1.9. You planned potassium replacement intravenously and passed a femoral central line. What is the maximum rate of potassium that can be given to this patient?
    A. 10 mEq per hour
    B. 20 mEq per hour
    C. 30 mEq per hour
    D. 40 mEq per hour
    E. 50 mEq per hour
    Ans: 40 mEq per hour

    Explanation: Through a peripheral line, maximum rate is 10 mEq per hour and through the central vein maximum 40 mEq per hour can be given under strict ECG monitoring. But ideally, 20 mEq per hour is used in less critical cases. Higher doses can cause cardiac arrhythmias and phlebitis because KCL is irritant to the veins. Maximum 24 hour replacement is 40 mEq for peripheral vein and 100 mEq for central vein.

25. Which of the following is not a cause of transcellular shift of potassium into the cells?
    A. Hypomagnesaemia
    B. Periodic paralysis
    C. Insulin infusion
    D. Metabolic alkalosis
    E. Albuterol nebulization
    Ans: Hypomagnesaemia
    Explanation: Magnesium depletion has inhibitory effects on muscle Na/K ATPase activity, reducing influx into muscle cells and causing a secondary urinary potassium loss. In addition, magnesium depletion causes exaggerated potassium secretion by the distal nephron and this effect is attributed to a reduction in the magnesium dependent intracellular block of potassium influx through the secretary potassium channel of principal cells. In consequence, hypomagnesemic patients are clinically refractory to potassium replacement in the absence of magnesium repletion. All the other causes are causes of transcellular shift of potassium with no net loss of potassium from the body.

26. Which of the following is the earliest ECG changes in hyperkalemia?
    A. Tall peaked T wave
    B. Short QT interval
    C. Prolonged PR interval
    D. QRS widening

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E. Absent P waves

Ans: Tall peaked T wave

Explanation: ECG changes have a sequential progression in hyperkalemia, which roughly correlate with the potassium level. Early changes of hyperkalemia include tall, peaked T waves with a narrow base, best seen in precordial leads. These changes are typically seen at a serum potassium level of 5.5-6.5 mEq/L. At a serum potassium level of 6.5-8.0 mEq/L, in addition to peaked T waves, the ECG shows the following:

- Prolonged PR interval
- Decreased or disappearing P wave
- Widening of the QRS
- Amplified R wave.

27. A 26 year old male presented in emergency with history of crush injury with decrease urine output. ECG done showed tall tented T waves with wide QRS complex. What should be the next best step in management?
    A. Check serum K
    B. Insulin infusion
    C. Calcium gluconate IV
    D. NAHCO3 IV
    E. Salbutamol nebulization

    Ans: Calcium gluconate IV

    Explanation: This patient with history of crush injury most probably having rhabdomyolysis causing typical ECG changes of hyperkalemia. The immediate step should be to stabilize the heart with IV calcium gluconate. Administer intravenous calcium to ameliorate cardiac toxicity. Infuse calcium chloride or calcium gluconate (10 mL of a 10% solution over 2-3 minutes). Onset of action occurs within minutes; duration of action is 30 minutes to an hour. All the other steps are also used after stabilizing heart.

28. A known case of CKD patient presented with complaints of muscle weakness and palpitations. Serum K is 6.8 mEq/L. ECG showed tall tented T waves. What is the best method to remove the potassium from the body in this patient?
    A. Calcium gluconate
    B. Insulin infusion
    C. NAHCO3 infusion
    D. Hemodialysis
    E. Furosemide Infusion

    Ans: Hemodialysis

    Explanation: The best initial step in treatment in this patient is calcium gluconate but the question asked about the best method to remove the potassium from the body. Calcium gluconate does not cause any change in the level of potassium in the body. Its main function is to stabilize the cardiac activity. Insulin infusion, salbutamol nebulization and NAHCO3 also cause transcellular shift of potassium and can be used initially in the management but these measures also do not cause change in total body potassium. Potassium excretion should be done after initial stabilization. There are two methods. GI excretion by using sodium polystyrene sulphonate, patiromer or sodium zirconium

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cyclosilicate, and hemodialysis. Hemodialysis is the best and most reliable method of removing potassium from the body and is the definitive treatment.

29. A 58 year old diabetic patient presented with complaints of muscle weakness with palpitations. Labs were done which showed creatinine 1.7, serum K 7.5, HCO3 15, Cl 115, Na 135 and pH 7.32. Urine sodium is 55 mmol/L. Blood pressure is 165/100. What is most probable diagnosis?

A. RTA type 1
B. RTA type 2
C. RTA type 4
D. Uremia
E. ACE inhibitors poisoning

Ans: RTA type 4

Explanation: The possible causes in this patient are either RTA type 4 or chronic kidney disease. This patient is having hyperkalemic non anion gap metabolic acidosis. Type 1 and 2 RTA are associated with hypokalemia. ACE inhibitors can cause hyperkalemia but acidosis is not present. Differentiation between CKD and RTA type 4 is done with measuring urinary sodium which would be low in case of CKD and high in case of RTA. Type 4 RTA commonly occurs in elderly diabetic patients and with mild to moderate CKD with hyperkalemia and acidosis out of proportion to impairment in GFR.

This is the most common form of RTA in adults and results from aldosterone deficiency or resistance. The collecting duct is a major site of aldosterone action; there it stimulates Na reabsorption and K secretion in the principal cells and stimulates H secretion in the A-type intercalated cells. Hypoaldosteronism, therefore, is associated with decreased collecting duct Na reabsorption, hyperkalemia, and metabolic acidosis. Hyperkalemia also reduces proximal tubular NH4+ production and decreases NH4+ absorption by the thick ascending limb, leading to a reduction in medullary interstitial NH3 concentration. This diminishes the ability of the kidneys to excrete an acid load and worsens the acidosis.

30. A 27 year old male presented in emergency after taking more than 50 tablets of digoxin. ECG was done which showed tall tented T waves with wide QRS complex and absent P waves. What should be the next best management in this patient?

A. Calcium gluconate
B. Calcium chloride
C. Magnesium sulphate
D. Insulin infusion
E. Hemodialysis

Ans: Magnesium sulphate

Explanation: This patient has typical ECG changes of hyperkalemia and digoxin toxicity can cause life threatening hyperkalemia. Hypercalcemia potentiates the cardiac toxicity of digoxin and should be avoided, instead, give magnesium sulfate (2 g over 5 minutes) for patients with cardiac arrhythmias from digitalis toxicity.

31. You received a call from gynecology department for a patient who was admitted with eclampsia. She was treated with magnesium sulphate. Patient has developed flaccid

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paraparesis and is confused. You ordered serum electrolytes which showed high serum magnesium. What should be the best initial treatment for her?

A. Urgent dialysis
B. Forced diuresis
C. Monitor only
D. Calcium chloride
E. Insulin infusion

Ans: Calcium chloride

Explanation: This patient has developed magnesium toxicity due to over treatment. Hypermagnesemia typically causes muscle weakness, hyporeflexia, confusion initially followed by flaccid paraparesis, ileus and respiratory muscle weakness in advance stages. Calcium antagonizes magnesium and calcium chloride should be given initially. Hemodialysis or peritoneal dialysis may be needed in case of CKD or poor kidney function.

32. A call is sent to you from ENT department for interpretation of abnormal blood tests for a diabetic patient who is admitted with mucormycosis, which showed Na 137, K 3.0, Cl 115 , HCO3 15, pH 7.31 and PaCO2 30mmhg. What can be the probable cause of abnormal blood tests in this patient?

A. Diabetic ketoacidosis
B. Acute kidney injury
C. Renal Tubular acidosis
D. Respiratory acidosis
E. Lactic acidosis

Ans: Renal tubular acidosis

Explanation: This patient is having normal anion gap metabolic acidosis with hypokalemia. The only cause from the above option which can cause normal anion gap metabolic acidosis is renal tubular acidosis. History also revealed that patient is diagnosed case of mucormycosis for which treatment of choice is amphotericin B. Amphotericin B is associated with development of distal (type 1) RTA. DKA, lactic acidosis and acute kidney injury cause high anion gap metabolic acidosis. This patient is having low CO2 level which suggests appropriate respiratory compensation for metabolic acidosis.

33. A 45 year old diabetic and hypertensive patient admitted with acute pulmonary edema. You have treated the patient with IV furosemide. Blood tests done the next morning are as follow: Na 134, K 3.6, Cl 100, HCO3 14, PaCO2 29, blood sugar random 150 and pH 7.31. What is the likely cause of abnormal results in this patient?

A. Diabetic ketoacidosis
B. Type 1 lactic acidosis
C. Type 2 lactic acidosis
D. Furosemide toxicity
E. Hyperventilation

Ans: Lactic acidosis type 1

Explanation: This patient is having high anion gap metabolic acidosis with appropriate respiratory compensation. DKA and lactic acidosis both can produce this type of picture, but with blood sugar of 150, DKA is less likely to be the cause. This patient presented with pulmonary edema may have low blood pressure, or due to hypoxia, there may be peripheral generation of lactate resulting in lactic

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acidosis. Type 1 lactic acidosis occurs due to tissue hypoxia and peripheral generation of lactate as in patients with circulatory failure and shock. Type 2 LA results due to impaired metabolism of lactate, as in liver disease or by a number of drugs and toxins, including metformin, which inhibit lactate metabolism. Furosemide therapy and toxicity is associated with metabolic alkalosis.

- If patient has metabolic acidosis, the next step is to calculate the anion gap. AG=Na – (HCO3+Cl)

34. A 36 year old female is found comatose, having taking some unknown number of pills. ABGs were done which showed pH 7.1, HCO3 12, PaCO2 72 mmhg and PaO2 65 mmhg. What is the interpretation of ABGs result?
    A. Compensated metabolic acidosis
    B. Compensated respiratory acidosis
    C. Mixed metabolic and respiratory acidosis
    D. Uncompensated metabolic acidosis
    E. Uncompensated respiratory acidosis

Ans: Mixed metabolic and respiratory acidosis

Explanation: This patient has low pH, high PaCO2 and low HCO3. In pure respiratory acidosis, Paco2 and HCO3 change in opposite direction to the change in pH i.e high Paco2 and high HCO3 with low pH. In pure metabolic acidosis, change in pH and change in HCO3 and PaCO2 are in the same direction i.e low pH, low HCO3 and low PaCO2. If the change is not like this, it is likely that more than one acid base disorder is present. This patient has mixed picture with low pH, high Paco2 and low HCO3, which suggest mixed respiratory and metabolic acidosis.

35. A 27 year old male admitted with Intestinal obstruction due to abdominal tuberculosis. ABGs were done by the surgical registrar and sent for interpretation. pH 7.55, Paco2 52 mmhg and HCO3 38. What is the underlying metabolic disorder?
    A. Respiratory alkalosis
    B. Metabolic acidosis
    C. Metabolic alkalosis
    D. Respiratory acidosis
    E. Mixed alkalosis

Ans: Metabolic alkalosis

Explanation: This patient has high pH, high HCO3 and high PaCO2 in a clinical context of Intestinal obstruction which causes vomiting and loss of gastric acid by NG aspiration. The primary disorder here is metabolic alkalosis with respiratory compensation. In primary metabolic alkalosis pH, HCO3 and PaCO2 go into same direction.

36. A 48 year old female was admitted with febrile gastroenteritis. Laboratory investigation showed pH 7.40, pco2 40, hco3 19, Na 144, K 2.9 and Cl 88. What is the acid base abnormality?
    A. Compensated normal AG metabolic acidosis
    B. Compensated high AG metabolic acidosis
    C. Metabolic acidosis with metabolic alkalosis
    D. Metabolic alkalosis
    E. Patient has no acid base disorder

Ans: Metabolic acidosis with metabolic alkalosis

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Explanation: The pH is normal in this patient. But anion gap is high (144-107=37) suggesting the presence of metabolic acidosis as a primary disorder. In patients with high anion gap metabolic acidosis, the increase in anion gap is equal to the decrease in HCO3 and the ratio of ΔAG/ΔHCO3 is equal to 1. When the increase in anion gap significantly exceeds the decrease in HCO3, a mixed picture is the most probable cause as in our patient.

ΔAG/ΔHCO3= 37-12/24-19=5

So, a superimposed metabolic alkalosis is suspected in this patient which is causing pH to be normal. This patient may have diarrhea associated with vomiting. Appropriate compensation tends to normalize the pH, but compensation does not cause normal pH.

- In high anion metabolic acidosis, the next step should be to calculate the ΔAG/ΔHCO3. If this ratio is 1-2, an uncomplicated metabolic acidosis is present. If < 1, then a concurrent non anion gap metabolic acidosis is likely to be present. If > 2, then a concurrent metabolic alkalosis is likely to be present.

37. A 68 year old male with a history of smoking presented with worsening SOB and increase in the production of sputum. His SaO2 at presentation is 84% on room air. ABGs were done which showed pH 7.25, PCO2 72, PO2 62 and HCO3 32. What is the diagnosis?
    A. Metabolic alkalosis with partial compensation
    B. Respiratory acidosis with compensation
    C. Mixed metabolic acidosis with respiratory acidosis
    D. Respiratory alkalosis
    E. Metabolic acidosis with metabolic alkalosis

    Ans: Respiratory acidosis with compensation

    Explanation: This patient has low pH (acidosis), high PCO2 (respiratory acidosis) and high HCO3 (metabolic alkalosis). In primary respiratory acid base problem, pH and change in PCO2 and HCO3 goes in opposite direction and in primary metabolic disorder, pH and change in PCO2 and HCO3 goes in same direction. So, here the combination of the low pH and high PCO2 tells us that the primary disorder is respiratory acidosis. The metabolic alkalosis is the compensatory process and pH is still low despite appropriate compensation which rules out concurrent metabolic alkalosis. Also history of smoking suggests acute exacerbation of COPD as a case which causes respiratory acidosis.

38. A 45 year old female is admitted with history of taking some unknown poison. She is having tachypnea with rapid shallow breathing. SaO2 is 94% on room air and ABGs were done which showed pH 7.01, PCO2 19, PO2 115, HCO3 5, Na 130, Cl 98 and K 4.1. What is the acid base disorder?
    A. High AG metabolic acidosis
    B. Normal AG metabolic acidosis
    C. Mixed high AG and normal AG metabolic acidosis
    D. Metabolic acidosis with metabolic alkalosis
    E. Respiratory alkalosis with compensation

    Ans: Mixed high AG and normal AG metabolic acidosis

    Explanation: This patient has very low pH (acidosis) with very low HCO3 (metabolic acidosis) associated with very low PACO2 (respiratory alkalosis). The low pH with low HCO3 suggests metabolic acidosis is the primary disorder and respiratory alkalosis is compensatory process. The next step is to

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calculate the anion gap which is 130- (98+5) = 27. So patient has high anion gap metabolic acidosis. The next step should be to calculate the delta gap. Delta gap = ΔAG/ΔHCO3= 27-15/24-5= 0.6. The delta gap is less than 1 which suggests that concurrent normal anion gap metabolic acidosis is also present.

39. A 22 year old female presented with sudden onset of shortness of breath. She is complaining of difficulty in taking deep breath. On examination, blood pressure is 110/70, pulse 105 with normal oxygen saturation. ABGs were done which showed pH 7.54, HCO3 23 and PCO2 22 with PaO2 155. What is the acid base disorder?
    A. Compensated metabolic acidosis
    B. Uncompensated respiratory alkalosis
    C. Compensated respiratory alkalosis
    D. Uncompensated metabolic alkalosis
    E. Respiratory alkalosis with Metabolic alkalosis

Ans: Uncompensated respiratory alkalosis
Explanation: This patient has high pH (alkalosis) with low PaCO2 (respiratory alkalosis). The high pH in conjunction with low PaCO2 tells us that respiratory alkalosis is the primary disorder. Although HCO3 is slightly low, but this patient has not mounted a compensatory response yet because metabolic compensation is slow to start than respiratory compensation. This patient most probably having hyperventilation due to panic attack.

40. A 26 year old male presented in emergency after taking some unknown quantity of an unknown drug. Patient is tachypneic at presentation with normal oxygen saturation. ABGs were done which showed pH 7.15, PCO2 13, PaO2 110, HCO3 10. What is the acid base abnormality?
    A. Compensated metabolic acidosis
    B. Compensated respiratory alkalosis
    C. Metabolic acidosis with respiratory alkalosis
    D. Uncompensated respiratory alkalosis
    E. Uncompensated metabolic acidosis

Ans: Metabolic acidosis with respiratory alkalosis
Explanation: This patient is having low pH (acidosis) with low HCO3 (metabolic acidosis). The combination of low pH and low HCO3 suggests metabolic acidosis as the primary disorder. The next step should be to check the compensation which in this case should be a respiratory compensation. In metabolic acidosis the expected compensation is calculated as PCO2= 1.5*ΔHCO3+8= 23. The PCO2 should be 21 to 25 with compensation. If it is less than this, a mixed disorder of respiratory alkalosis will be present and if it is more than 25 in this patient, concurrent respiratory acidosis would be present. In our patient, PCO2 is only 13 which suggests that respiratory alkalosis is also present. This is a common metabolic abnormality in aspirin poisoning which causes metabolic acidosis with respiratory alkalosis.

41. Which of the following test is used to differentiate between type 1 and type 2 RTA?
    A. Serum potassium
    B. Serum pH
    C. Urinary bicarbonate
    D. Urine sodium
    E. Anion gap

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Ans: Urinary bicarbonate

Explanation: Two important tests used to differentiate between type 1 and type 2 RTA are urinary pH and urinary bicarbonate level or fractional excretion of bicarbonate. In type 1 RTA, the defect is in distal acid secretion, leading to gain of H ion which leads to acidosis with less acidic urine. Type 1 RTA should be suspected in any patient with non-AG metabolic acidosis and a urine pH greater than 5.0. Patients have a reduction in serum HCO3 to various degrees, in some cases to less than 10 mEq/L. They are able to reabsorb HCO3 normally, and their fractional excretion of HCO3 is less than 3%. The hallmark of type 2 RTA is impairment in proximal tubular HCO3 reabsorption. Patients with type 2 RTA have a lower threshold for excretion of HCO3, leading to a loss of filtered HCO3. The diagnosis of type 2 RTA should be suspected in patients who have a normal-AG metabolic acidosis with a serum HCO3 level usually greater than 15 mEq/L and acidic urine (pH < 5.0). Fractional excretion of bicarbonate is usually more than 15% in RTA type 2.
Serum potassium is used to differentiate type 1 RTA (hypokalemia) from type 4 RTA (Hyperkalemia).

42. Which of the following is not a cause of metabolic alkalosis?
    A. Gitelman syndrome
    B. Bartter syndrome
    C. Liddle syndrome
    D. Fanconi syndrome
    E. Vomiting

Ans: Fanconi syndrome

Explanation: All of the above are the causes of metabolic alkalosis except Fanconi syndrome, which is associated with normal AG metabolic acidosis. It causes generalized defect of the proximal tubule characterized by glucosuria, bicarbonaturia, hypokalemia, metabolic acidosis, aminoaciduria, and phosphaturia.

43. A 37-year-old woman is referred to you after two episodes of nephrolithiasis. On questioning she reports that pain and redness develop in her hands in cold weather. She takes no medications except for occasional antibiotics for her sinus problems. Her BP is 108/50 mmHg and her physical examination is unremarkable except for some fullness over her parotid glands. Her labs were done which showed Na 141, K 2.9, Cl 125, HCO3 12, Ca 9.9 and mg 2.1. What is the most likely diagnosis?
    A. Type 1 RTA
    B. Type 2 RTA
    C. Type 4 RTA
    D. Milk alkali syndrome
    E. Gitelman syndrome

Ans: Type 1 RTA

Explanation: This patient presents with a non–anion gap metabolic acidosis along with hypokalemia. Because her history is also notable for symptoms suggesting a collagen vascular disorder (parotid gland swelling, Raynaud phenomena) and there is no mention of significant diarrhea, distal renal tubular acidosis associated with Sjögren syndrome should be suspected and appropriate serologic studies should be obtained. Type 1 RTA is also associated with nephrocalcinosis and increased risk of calcium phosphate stones formation. In the presence of metabolic acidosis, the bone serves as an important buffer and exchanges H+ ions for sodium, calcium, and potassium that are complexed to

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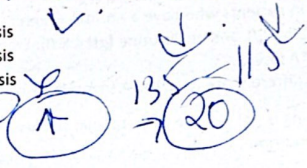
carbonates and phosphates. Over time, this leads to increased bone resorption with increased calcium and phosphate release and hypercalciuria with hyperphosphaturia. Compounding this is that metabolic acidosis leads to increased citrate resorption in the proximal tubule and a decrease in urine citrate, a prime inhibitor of stone formation. Finally, the alkaline pH of the urine fosters precipitation of calcium phosphate, leading to stones and/or nephrocalcinosis.

44. A 30 year old woman with a history of alcoholism presents with altered sensorium, rapidly rising hepatic enzymes and the following laboratory data: serum creatinine 1.5, Glucose 102, Na 135 mEq/L; potassium 4.9 mEq/L; bicarbonate 15 mEq/L and chloride 100 mEq/L. The arterial pH is 7.32 and the PaCo2 29. Blood ketones and blood alcohol were negative. The diagnosis is:
    A. Type 1 lactic acidosis
    B. Type 2 lactic acidosis
    C. Diabetic ketoacidosis
    D. Ethanol poisoning
    E. Uremia



Ans: Type 2 lactic acidosis

Explanation: This patient is having high anion metabolic acidosis. According to history, the most probable diagnosis is type 2 lactic acidosis.

45. Fanconi syndrome is a type 2 renal tubular acidosis along with aminoaciduria, glycosuria and phosphaturia. Which of the following is also a presenting feature of Fanconi syndrome?
    A. Nephrocalcinosis
    B. Nephrolithiasis
    C. Osteomalacia
    D. Anemia
    E. Thrombocytopenia

Ans: Osteomalacia

Explanation: Due to the loss of excessive calcium and phosphate and a defect in the hydroxylation of 25 hydroxyvitamin D, Osteomalacia is a common presenting feature of Fanconi syndrome in adults. Nephrocalcinosis and nephrolithiasis are features of distal RTA. Anemia is associated with Fanconi anemia which is a different disorder and most common cause of congenital Aplastic anemia.

46. A 50 year old patient is taken to emergency by 1122 after found him on road in a comatose state. His labs were done which showed Na 150, K 3.1, Cl 105, HCO3 10, pH 7.21, PCO2 25, glucose 55 and BUN 40. His measured serum osmolality is 375. What is the most probable cause of this patient presentation?
    A. Non hyperglycemic diabetic ketoacidosis
    B. Type 1 lactic acidosis
    C. Alcohol poisoning
    D. Uremic encephalopathy
    E. Type 2 Lactic acidosis

Ans: Alcohol poisoning

Explanation: This patient is having low pH with low HCO3 which suggest metabolic acidosis. The low PCO2 is respiratory compensation. The next step is to calculate anion gap which is 35 in this case which

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shows high AG metabolic acidosis. To narrow down the differentials of high AG metabolic acidosis, next step should be to calculate the osmolal gap as follow:

Osmolal gap= measured osmolality- calculated osmolality. It should be less than 10. A large positive osmolar gap can help identify the presence in plasma of substances such as ethanol, methanol, isopropanol, ethylene glycol and propylene glycol. In our patient, the osmolal gap is almost 55 which showed very large osmolal gap and suggests poisoning by any of the above poison. All the other causes are normal osmolal high anion gap metabolic acidosis except alcohol in our patient.

47. A 26 year old female with history of migraine is presented with general body weakness and muscles aches and pains. Labs were done which showed Na 132, K 3.1, Cl 110, HCO3 14, pH 7.29 with urine pH 5.1. What is the most probable cause of abnormal blood tests in this patient?
    A. Naproxen
    B. Sumatriptan
    C. Topiramte
    D. Propranolol
    E. Ergotamine

Ans: Topiramte

Explanation: This patient has hypokalemic non AG metabolic acidosis with acidic urine which suggests renal Tubular acidosis as the cause. Topiramte can cause mixed type 1 and type 2 RTA. It does this by impairing both the normal reabsorption of filtered HCO3– by the proximal renal tubule and the excretion of H+ by the distal renal tubule. This combination of defects is termed mixed renal tubular acidosis (RTA). The mechanism involves the inhibition of the enzyme carbonic anhydrase, which is consistent with the fact that genetic deficiency of carbonic anhydrase is associated with mixed RTA. Topiramate-induced RTA can make patients acutely ill, and chronically, can lead to nephrolithiasis, osteoporosis.

48. A 65 year old male is diagnosed as having gastric outlet obstruction on endoscopy. Patient has history of intractable vomiting for 2 months. What is the most probable metabolic abnormality in this patient?
    A. pH 7.31, HCO3 18, aciduria
    B. pH 7.55, HCO3 35, alkalinuria
    C. pH 7.55, HCO3 35, aciduria
    D. pH 7.30, HCO3 15, alkalinuria
    E. pH 7.55, HCO3 15, alkalinuria

Ans: pH 7.55, HCO3 35, aciduria

Explanation: Patients with gastric outlet obstruction have unique metabolic sequelae, namely hypochloraemic, hypokalaemic metabolic alkalosis with paradoxical aciduria and hypocalcaemia. The pathophysiology of this biochemical abnormality is persistent vomiting of gastric hydrochloric acid resulting in hypochloraemia and metabolic alkalosis. As dehydration progresses, metabolic abnormalities become more profound and renal function is overwhelmed. Initially, the urine has low chloride and high bicarbonate content to compensate for gastric losses of hydrochloric acid and is appropriately alkaline. As a result of continuous dehydration, sodium gets reabsorbed preferentially over potassium and hydrogen ions that are excreted by kidneys because the first response of body is always to correct the ECF volume. Decreased ECF volume activates RAAS which

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results in sodium and water reabsorption with loss of K and H. This results in urine becoming paradoxically acidic and hypokalemia ensues. Alkalosis leads to a reduction of the circulating ionized calcium thereby completing the biochemical syndrome.

5 step approach for interpretation of ABGs:

Step 1: Check the pH. Is there alkalemia or acidemia present?

If pH < 7.35 => acidemia

If pH > 7.45 = > alkalemia

This is usually a primary disorder, pH can be normal with mixed opposite disorders for which you will need PCO2, HCO3 and anion gap

Step 2: Is the disturbance respiratory or metabolic? What is the relationship between the direction of change in the pH and the direction of change in the PaCO2? In primary respiratory disorders, the pH and PaCO2 change in opposite directions; in metabolic disorders the pH and PaCO2 change in the same direction.

Acidosis	Respiratory	pH low	CO2 high	High HCO3 in compensation
Acidosis	Metabolic	pH low	HCO3 low	Low PCO2 in compensation
Alkalosis	Respiratory	pH high	Co2 low	Low HCO3 in compensation
Alkalosis	Metabolic	pH high	HCO3 high	High PCO2 in compensation

Step 3. Is there appropriate compensation for the primary disturbance? Usually compensation does not return the pH to normal.

Disorder	Expected compensation
Metabolic acidosis	PCO2= 1.5*ΔHCO3+8
Acute respiratory acidosis	Increase in HCO3= ΔPCO2/10
Chronic respiratory acidosis	Increase in HCO3= 3.5(ΔPCO2/10)
Metabolic alkalosis	Increase in PCO2= 40+0.6(ΔHCO3)
Acute respiratory alkalosis	Decrease in HCO3= 2(ΔPCO2/10)
Chronic respiratory alkalosis	Decrease in HCO3 = 5(ΔPCO2/10)

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If the observed compensation is not the expected compensation, it is likely that more than one acid base disorder is present.

Step 4. Calculate the anion gap (if a metabolic acidosis exists): AG= Na-(Cl- + HCO3-)

- Normal anion gap is approximately 12 meq/L.
- In patients with hypoalbuminemia, the normal anion gap is lower than 12 meq/L; the "normal" anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration.
- If the anion gap is elevated, consider calculating the osmolal gap in compatible clinical situations. It is used to narrow down the differentials of high anion gap metabolic acidosis. OSM gap = measured OSM – (2[Na+] - glucose/18 – BUN/2.8. The osmolal gap should be less than 10 in common causes like DKA, Uremia and lactic acidosis. High osmolal gap is present in ethanol, methanol, ethylene glycol and isopropyl alcohol poisoning.

Step 5: If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in HCO3. Assess the ratio of the change in the anion gap (ΔAG) to the change in HCO3 (ΔHCO3): ΔAG/ΔHCO3.

- This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present.
- If this ratio falls outside of this range, then another metabolic disorder is present. If ΔAG/ΔHCO3 < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present. If ΔAG/ΔHCO3> 2.0, then a concurrent metabolic alkalosis is likely to be present.

Mixed and complex acid base disorders:

R. Acidosis+ M. acidosis	Low pH, low HCO3, high PCO2	Cardiac arrest, intoxication, multiorgan failure
R. Alkalosis+ M. Alkalosis	High pH, high HCO3, low PCO2	Cirrhosis with diuretics, vomiting in pregnancy, over ventilation in COPD
R. Acidosis + M. Alkalosis	pH normal, PCO2 high, HCO3 high	COPD with diuretics, vomiting or NG aspiration, severe hypokalemia
R. Alkalosis + M. Acidosis	pH normal, HCO3	Salicylate poisoning, renal failure

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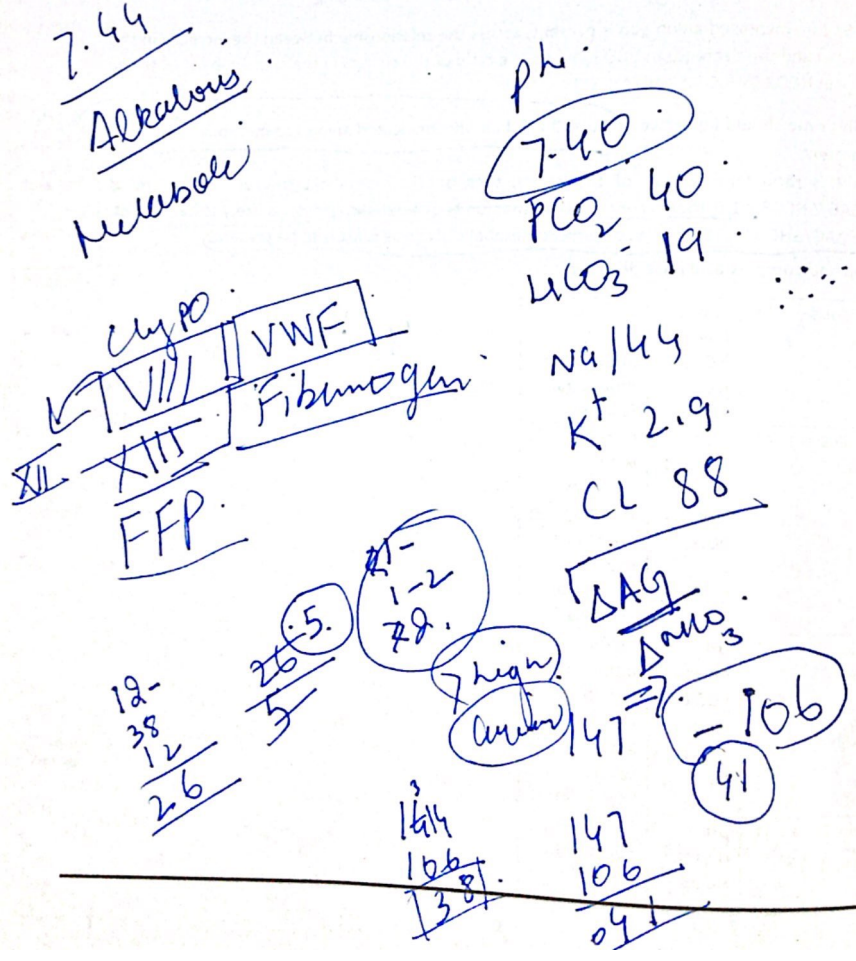
	low, PCO2 high	with pneumonia, sepsis.
M. Acidosis+m.alkalosis	pH normal, HCO3 normal, PCO2 normal, increase delta gap	Uremia or DKA with vomiting or diuretics



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F-aget sign

Typhoid

appendix (abscess)

Strept (sore throat)

Dental

penicillin if allergic

Clindamycin

Odontogenic

Oxacillin then

Levo + Refampin

Gram